Status Epilepticus

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KEYWORDS

• Status epilepticus • Seizure • Benzodiazepine • Pathophysiology
• Nonconvulsive status epilepticus • Antiseizure medications • Neuronal injury
• Refractory status epilepticus

KEY POINTS

- Status epilepticus (SE) is defined as prolonged seizure activity, or recurrent seizure activity without a return to baseline.
- SE can involve many types of seizure activity, from tonic-clonic activity to nonconvulsive status epilepticus. EEG monitoring is essential for diagnosis of nonconvulsive SE.
- Status epilepticus can lead to permanent neuronal injury and is associated with pharmacoresistance. Recognition of SE should be immediately followed by appropriately-dosed benzodiazepines.
- Clinicians should carefully assess the seizing patient's airway and be sure to check a point-of-care glucose in all patients and pregnancy status in reproductive-age females.
- Clinicians should be familiar with the stepwise approach to treating SE, first using benzodiazepines, then using antiseizure medications, and then finally intubation with anesthetic medications.

INTRODUCTION

Status epilepticus (SE) is a neurologic emergency that carries significant mortality and morbidity for patients and poses many management challenges for clinicians. The approach to SE, including the definitions, treatment, and understanding of pathophysiology have significantly evolved over the past decade, necessitating that emergency physicians be familiar with up-to-date guidelines and advances to provide the best care for these patients.

DEFINITIONS

Status Epilepticus

SE refers to prolonged or recurrent episodes of seizure-like activity without recovery. Previously, this definition consisted of either continuous seizure-like activity or the

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Abbreviations	
BZDs	benzodiazepines
CNS	central nervous system
EEG	electroencephalography
ESETT	established status epilepticus treatment trial
GABA	γ-aminobutyric acid
IM	intramuscular
IN	intranasal
IV	intravenous
NCSE	nonconvulsive status epilepticus
NMDA	N-methyl-D-aspartate
NORSE	New-onset refractory status epilepticus
PNEA	psychogenic nonepileptic activity
SE	status epilepticus

occurrence of at least 2 episodes of seizure-like activity without the recovery of consciousness lasting beyond 30 min.[1] In 2015, the International League Against Epilepsy proposed to decrease the time to 5 min of continuous convulsive seizure-like activity or 10 min of continuous focal seizures with impaired consciousness, and 10 to 15 min for absence seizures.[2] After this time point, the patient is considered to be in SE, and treatment should be initiated immediately as the likelihood of spontaneous cessation of such seizure-like activity is low. The rationale behind this change in definition (and subsequently, change in management), is that neuronal damage and long-term, potentially irreversible consequences of prolonged seizure activity occurs at 30 min of convulsive seizure activity and 60 min of focal SE.[2] The Abnormal speech, ocular Deviation, Automatisms, and Number of motor epileptic seizures scale, or ADAN scale, has been developed for detecting patients at high risk for SE with a 91% predictive power, and can be used to facilitate early identification of SE.[3]

Refractory and Super-refractory Status Epilepticus

Refractory SE is defined as SE that persists after the administration of 2 antiseizure medications, while super-refractory SE is defined as SE that persists beyond 24 hours after the initiation of anesthesia, or recurs on weaning of anesthesia.[2]

EPIDEMIOLOGY

SE affects from 10 to 41 per 100,000 people annually, with a slightly lower incidence in Europe than in the United States.[4–7] Among Americans, there is a higher incidence among African American people than in White people, who in turn have a higher incidence than in Asian and Hispanic people.[8] Approximately 50,000 to 60,000 new cases of SE are diagnosed annually in the United States.[4,9] More than half the first episodes of SE occurred in the absence of a known diagnosis of epilepsy.[4] New-onset refractory SE (NORSE) is a subtype of it as well,[2] with one study finding up to 45.6% of cases of new onset SE progressing to NORSE.[10] When SE is associated with a diagnosis of epilepsy, it usually occurs relatively early in the course of epilepsy, often representing the first or second seizure in 65% of cases. Patients who have experienced an episode of SE have a 3.3 times higher risk of subsequent unprovoked seizures when compared to those who have experienced a single, self-limited symptomatic seizure.[4] The incidence of SE appears to have a bimodal age distribution, with peaks in patients less than 10 year old and greater than 50 years old.[11] Critically ill patients are particularly susceptible to SE, with more than 50% of patients with generalized convulsive SE having a history of acute brain injury (such as strokes, trauma, or anoxia) or acute

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systemic processes (including infections, hypoglycemia, or substance intoxication or withdrawal).[12]

Morbidity and Mortality

SE can carry a significant mortality rate; among adults, the 30-day mortality rate is 10.2%, while the 1-year mortality is 25.1%.[13] The mortality of SE is directly proportional to how refractory it is to therapy.[14] Among patients with refractory SE, including super-refractory SE, it is estimated that roughly one-third of patients will die, one-third will survive with mild or severe neurologic deficits, and one-third will recover to their baseline.[15]

PATHOPHYSIOLOGY

The pathophysiology of SE, while not entirely elucidated, revolves around a failure of the body to endogenously terminate a seizure either because of excess excitation or of a loss of inhibitory mechanisms, ultimately allowing what may begin as a single seizure to persist into SE. The onset of a seizure immediately triggers a multitude of events within milliseconds–from neurotransmitter release to ion channel aperture and closure to protein phosphorylation. Within seconds to minutes, there is an endocytosis-mediated decrease in the number of inhibitory γ-Aminobutyric acid (GABA) receptors, coupled with a concomitant increase in the amount of excitatory glutamate and N-methyl-D-aspartate (NMDA) receptors. Minutes to hours after the onset of the seizure, there is an increase in excitatory neuropeptides and a decrease in inhibitory neuropeptides, maintaining a state of increased excitability. Changes in gene expression begin to set in over the next hours to weeks, potentially contributing to increased susceptibility to future seizure activity.[16]

The excessive neuronal firing during seizures has been shown to contribute to neuronal death through a variety of mechanisms including apoptosis, necrosis, and mitochondrial dysfunction. This neuronal injury was observed to occur even during paralysis, indicating that both convulsive and nonconvulsive SE (NCSE) can contribute to neuronal death. Furthermore, seizures have been found to contribute to hyperthermia, metabolic acidosis, hypotension, and hypoxia, all of which can also contribute to cell death in multiple regions of the brain.[17]

The mechanisms that lead to the development of SE are also believed to contribute to the pharmacoresistance that is seen in SE. The downregulation of GABA receptors leads to decreased efficacy of GABA-agonist medications such as benzodiazepines in controlling seizure activity. This pharmacoresistance underscores the importance of early recognition of SE and prompt initiation of treatment to prevent decreasing efficacy of antiseizure medications, further illustrating that *time is brain*.

CLASSIFICATION

Semiology

Semiology refers to the study of the symptoms and signs that occur during an epileptic seizure. SE can be categorized into SE with prominent motor symptoms and SE without prominent motor symptoms,[2] with the caveat that the clinical symptomatology can vary throughout a patient's course.[18] Motor manifestations can include generalized tonic-clonic (convulsive), myoclonic, tonic, focal motor, and hyperkinetic SE. The majority of seizure-like activity involved in SE is generalized tonic-clonic seizures.[19] Myoclonic SE consists of frequent, involuntary muscle contractions affecting 1 or more muscle groups. Tonic SE involves a persistent tonic posture and is more common in syndromic epilepsy such as Lennox-Gastaut syndrome. Focal motor SE has varying clinical features depending on the area of the cortex affected and can

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often involve multiple areas of focal activity as the seizure progresses.[2] NCSE does not feature any motor phenomena and will be discussed in a later section.

Etiology

The cause of SE is important to determine the appropriate treatment and prognosis and may be known (symptomatic) or unknown (cryptogenic). The known causes are summarized in **Box 1**, noting that the list is not exhaustive. The most common etiology of SE is acute symptomatic (58.8%), and within this category, the most common causes are primary and secondary central nervous system (CNS) insults.[20] It is important to remember that the cause of SE is not the same as the cause of the underlying disease.[2]

RESUSCITATION OF PATIENTS IN STATUS EPILEPTICUS

As with any medical emergency, the fundamentals of managing the airway, breathing, circulation, and disability of patients in SE are a priority. Immediate assessment of the patient's ABCs and continuous vital sign monitoring should occur in any patient presenting with seizure activity. Intravenous (IV) access should also be obtained as quickly and safely as possible. A point-of-care blood glucose should be obtained in

Box 1
Known causes of status epilepticus[2]

Known (Symptomatic)

Acute
- Triggering factor in epilepsy
  - Withdrawal of antiseizure medications
  - Febrile illness
  - Sleep deprivation
- Primary CNS pathology
  - Cerebrovascular disease
  - CNS infection
  - Head trauma
  - Autoimmune disorder
- Secondary CNS pathology
  - Metabolic disturbance (eg, electrolytes, glucose, acidosis, and organ failure)
  - Systemic infection
  - Fever
- Toxic causes
  - Intoxication by drug(s) or alcohol
  - Alcohol withdrawal

Remote
- Post-traumatic head injury
- Post-infectious
- Post-stroke

Progressive
- Brain tumors
- Progressive myoclonic epilepsies
- Neurodegenerative disorders

SE in defined electroclinical syndromes

From Luis Restrepo-Vera J, Sala-Padró J, Parejo-Carbonell B, et al. Identifying risk for status epilepticus with the ADAN scale: a prospective multicenter validation study. Emerg Rev Soc Espanola Med Emerg 2024;36(3):197–03. https://doi.org/10.55633/s3me/020.2024.

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any patient presenting with seizure-like activity, including altered mental status, and if found to be hypoglycemic, they should receive immediate dextrose.

In addition to hypoglycemia, another important cause of SE that must be quickly excluded is hyponatremia. Neurologic symptoms, including seizures, are typically not seen until sodium concentrations approach 120 mEq/L and below.[21] Point-of-care electrolyte devices, if available, can provide rapid results on sodium concentration. If the seizing patient is determined to be significantly hyponatremic, they should receive between 100 and 150 mL of 3% hypertonic saline or 2 ampules of sodium bicarbonate, both of which provide an equivalent amount of sodium load, over 10 minutes. If symptoms do not improve with an initial bolus of hypertonic saline, this bolus may be repeated up to 2 times; if seizures continue to persist, clinicians should consider other etiologies of patient's seizures. The goal is to raise the sodium 4 to 6 mEq/L in the first few hours, and to not exceed 10 to 12 mEq/L in the first 24 hours. Frequent electrolyte monitoring is necessary in these patients to avoid overcorrection of sodium levels and precipitation of osmotic demyelination syndrome (also known as central pontine myelinolysis).[22]

AIRWAY MANAGEMENT AND INTUBATION

Airway management in patients with SE is of vital importance. Patients in tonic-clonic seizures may display periods of apnea and cyanosis, with suppression of the gag reflex and risk of aspiration. Patients who are seizing should have an airway adjunct (such as nasopharyngeal or oropharyngeal airways) placed, receive supplementary oxygen to support their oxygenation, and be placed in the lateral decubitus position to decrease the risk of aspiration. Ventilatory assistance with a bag-valve-mask device should be administered if apnea or airway compromise is evident. The risk of airway and ventilatory compromise increases the longer a patient is actively seizing. Patients with treated SE are less likely to have respiratory complications than those who are untreated.[23] Therefore, appropriately dosing medications to manage the SE is necessary to decrease the risk of respiratory complications. Fifteen to thirty percent of SE patients undergo intubation, which has been associated with a higher mortality.[24,25] While etomidate is an often-used induction agent, it may not be the optimal agent in this patient population. Other induction agents, such as benzodiazepines (BZDs), propofol, and ketamine, have known antiseizure properties, which make them preferable in the intubation of the SE patient, significantly decreasing the time to cessation of seizure activity as measured by electroencephalography (EEG) when compared to etomidate.[26] Moreover, etomidate is commonly associated with myoclonic activity that may be confused with seizure-like activity, possibly complicating the clinical picture.[27] However, it does not decrease the seizure threshold as previously postulated.[28] Although propofol and BZDs have well-known antiseizure properties, ketamine may be a preferable induction agent given its hemodynamic stability. Once stabilized, propofol may be then used for sedation as well as for its antiseizure effects.[29] Although the use of a combination of lower doses of propofol and ketamine has been proposed for induction in patients with SE, this has not yet been established as a validated practice.[30]

With regards to paralytic agents, there is no evidence that supports the use of nondepolarizing (eg, rocuronium) versus depolarizing neuromuscular blockers (eg, succinylcholine). Clinicians should exercise judgment over which neuromuscular blockade agent to use and consider factors including comorbidities, duration of seizure activity, availability of reversal agents for nondepolarizing agents, as well as the ability to monitor continued seizure activity using an EEG.

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DIAGNOSIS

The differential diagnosis of convulsive SE includes psychogenic nonepileptic activity (PNEA), movement disorders (eg, myoclonus), and syncope. Although laboratory testing may help differentiate between these causes, the diagnosis of SE in the emergency department is largely clinical, depending on the history and physical examination. Two laboratory tests that may help with this differentiation are serum prolactin and lactic acid levels. Elevated prolactin levels, measured 10 to 20 min after a seizure event, has 99.1% specificity and 53% sensitivity for SE, differentiating it from PNEA.[31] In addition, lactic acid levels are significantly higher in patients with convulsive SE compared to those with PNEA or syncope.[32]

The diagnostic workup is important in identifying the underlying cause of the SE, if present (**Box 1**). Laboratory testing should include a complete blood count, glucose and electrolytes, and a pregnancy test in patients with childbearing potential, at a minimum.[33] Additional testing for renal and liver function tests, blood gas, lactate, creatine kinase, and toxicology screening may be added. Drug levels for certain antiseizure medications may be helpful in patients with known seizure disorders.[34] An infectious workup, including a lumbar puncture, should be guided by the clinical picture. A lumbar puncture is indicated in patients with signs of meningitis, those who are ill appearing, or have a fever with no other identifiable source of infection, especially if they had received antimicrobials before clinical evaluation. It is worth noting that any febrile illness can precipitate SE, especially in patients with known seizure disorder. In addition to uncovering an infectious cause, a lumbar puncture may identify inflammatory or neoplastic causes of the SE. CT head imaging should be performed in patients who do not return to baseline level of consciousness, have new focal neurologic findings, or have new-onset seizure without an otherwise identifiable etiology.[34] It should also be performed before the lumbar puncture in patients suspected to have increased intracranial pressure or a space-occupying lesion.

MANAGEMENT

Given the time-sensitive nature of treating SE due to neuronal injury and pharmacoresistance, clinicians should have a stepwise approach for management of patients in SE once it is recognized. Having an established hospital protocol to manage SE may significantly decrease time to treatment and improve outcomes.[35,36] In addition to the management principles outlined later, the underlying cause of seizures should be promptly treated, if known.

First-Line Treatment

The mainstay of treatment of patients in SE is prompt administration of adequately-dosed BZDs. The mechanism of action of BZDs involves agonism of GABA receptors through increasing the frequency of chloride channel opening on the receptors. Multiple prehospital studies have validated the efficacy and safety of BZDs in aborting seizure activity. In a prehospital study by Alldredge and colleagues[23] comparing lorazepam, diazepam, and placebo, 59.1% and 42.6% of seizures were aborted by the administration of lorazepam and diazepam, respectively, compared to 21.1% of seizures in patients given placebo.

Data is unclear regarding the preferred BZD to be used in SE; intramuscular (IM) midazolam, IV lorazepam, and IV diazepam are all efficacious as initial therapy (Level A recommendation, American Epilepsy Society Guideline 2020).[37] The time of administration of BZDs–specifically as it relates to the time from the start of

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the seizure–appears to be a more important factor than the route or specific agent. The Rapid Anticonvulsant Medication Prior to Arrival Trial noninferiority prehospital study compared IV lorazepam to IM midazolam, finding that IM midazolam was superior in the termination of seizures. Although IV lorazepam was associated with faster termination of convulsive activity from the time of administration than IM midazolam (1.6 vs 3.3 min), seizures were aborted in 73.4% of patients in the IM midazolam group versus 63.4% of those in the IV lorazepam group. This is most likely due to a shorter time to administration in the IM midazolam group than the IV lorazepam (1.2 vs 4.8 min).[38] These studies underscore the importance of early treatment of SE with BZDs, and solidify the superior effectiveness of IM midazolam in comparison to IV lorazepam in patients without established IV access. (Level A recommendation, American Epilepsy Society Guideline 2020).[37] Diazepam is another BZD that may be administered IV with demonstrated efficacy in terminating seizure activity; while lorazepam has been found to have a longer duration of action,[39] literature has not demonstrated any benefit of lorazepam over diazepam in treating SE.[40] Other potential options for administration of BZDs include buccal or intranasal (IN) midazolam, as well as rectal diazepam when appropriate. IM administration of lorazepam, while associated with lower bioavailability and slower absorption, remains an option should no alternative exist.[41] Familiarity with the local options is necessary, given the variable availability due to cost, drug shortages, and storage mechanisms. For ongoing seizure activity, the dose may be repeated once 5 minutes after the initial dose.[42] The appropriate doses are summarized in **Table 1**.

Despite robust data scoring their effectiveness and safety, BZDs continue to be underdosed. In the Established Status Epilepticus Treatment Trial (ESETT), lorazepam and midazolam were appropriately dosed in only 24% and 14% of studied patients, respectively.[43] It has been postulated that much of the underdosing of BZDs stems from concern over depression of the patient's respiratory drive. However, appropriately dosed treatment of seizing patients is associated with lower rates of respiratory depression.[23,37] Upon recognition of SE, the patient should receive appropriate doses of BZDs in the most expeditious way possible, whether IM, IN, or IV. When BZDs are not available, phenobarbital may be given as IV or IM.[37]

Second-Line Treatment

Up to one-third of patients in SE who are treated with BZDs will continue to display seizure activity, termed *established SE*,[2] necessitating the administration of additional IV antiseizure medications. Second line agents include phenytoin or fosphenytoin, valproate, and levetiracetam. These agents modulate excitatory activity near the synaptic cleft, with many of these drugs inhibiting excitatory channels within the presynaptic neuron or, with levetiracetam, blocking the release of excitatory glutamate into the synapse. Research has not demonstrated a clear advantage of any of these agents, with the ESETT study finding no significant difference in terminating benzodiazepine-refractory SE between levetiracetam, fosphenytoin, and valproate, when given at the appropriate weight-based doses as noted in **Table 1**.[43] Any of these medications may be administered in an SE patient who is not responding to BZDs (Level A recommendation),[44] within 20 to 40 min of SE onset.[37] When both fosphenytoin and phenytoin are available, fosphenytoin is preferred due to improved tolerability and faster infusion rate, but phenytoin is an acceptable alternative (Level A recommendation, American Epilepsy Society Guideline 2020).[37] An important consideration amongst these medications is the rate of administration; phenytoin is the slowest medication to administer. Administering the loading dose

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Table 1
Antiseizure dosages[8,43]

Medication	Dosage	Maximum Dose
First-line treatment		
Diazepam	0.2 mg/kg	10 mg
Lorazepam	0.1 mg/kg	4 mg
Midazolam	0.3–0.5 mg/kg	10 mg
Second-line treatment		
Phenytoin	20 mg/kg	1500 mg
Fosphenytoin	20 mgPE/kg	1500 mgPE
Valproic acid	40 mg/kg An additional dose of 20 mg/kg may be administered	3000 mg
Levetiracetam	60 mg/kg	4500 mg
Third-line treatment		
Lacosamide	200–400 mg IV	30–40 mg/min
Phenobarbital	15–20 mg/kg	60 mg/min
Anesthetics		
Ketamine	*Bolus:* 0.5–3 mg/kg *Infusion:* 1.5–10 mg/kg/h	
Midazolam infusion	*Bolus*: 0.2 mg/kg, repeated every 3–5 min to a max of 2 mg/kg *Infusion*: 0.05–2 mg/kg/h	
Propofol infusion	*Bolus*: 1–5 mg/kg, repeated in 3–5 min *Infusion*: 20–200 mcg/kg/min	
Pentobarbital infusion	*Bolus*: 5 mg/kg, repeated q3-5 min to a max of 15 mg/kg over 1 h *Infusion*: 0.5-5 mg/kg/h	
Thiopental infusion	*Bolus:* 1–5 mg/kg *Infusion:* 0.5–5 mg/kg/h	

Abbreviations: PE, phenytoin-equivalent.

Adapted from Luis Restrepo-Vera J, Sala-Padró J, Parejo-Carbonell B, et al. Identifying risk for status epilepticus with the ADAN scale: a prospective multicenter validation study. Emerg Rev Soc Espanola Med Emerg. 2024;36(3):197-203. doi:10.55633/s3me/020.2024.[8,43]

of undiluted levetiracetam as an IV push appears to be safe and tolerable,[45] making this an important advantage of levetiracetam. The use of a patient's home medication as the second-line agent does not appear to affect the probability of stopping the seizure.[46]

Third-Line Treatment

Up to one-half of patients in SE who are treated with a second-line agent will continue to display seizure activity.[47] The patient may be given another second-line medication, or a different antiseizure medication. A meta-analysis of 5 randomized controlled trials comparing valproate, phenytoin, diazepam, phenobarbital, lacosamide, and

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levetiracetam found that phenobarbital performed best in terminating seizure activity but was associated with a higher incidence of adverse effects such as hypotension and respiratory depression. However, the dosages noted in the study were inconsistent, affecting the accuracy of the results.[48]

Anesthetics

When the patient has received BZDs and 2 second-line treatments for SE and continues to exhibit seizure activity, the next appropriate step would be anesthetics and intubation. Some experts recommend a more rapid progression to IV anesthetics and intubation after the administration of only one second-line agent, given anesthetics' association with faster cessation of SE and higher likelihood of return to baseline, minimizing neuronal injury and pharmacoresistance.[8,49–51] While no drug has emerged as the ideal anesthetic infusion for combatting super-refractory SE, the most commonly used medications for anesthesia induction include midazolam and propofol, both of which have been found to have similar efficacy and incidence of adverse effects.[36] Other anesthetic infusions include pentobarbital and thiopental. All these anesthetic infusions have been associated with various side effects, including hypotension that necessitates the use of vasopressors such as norepinephrine, phenylephrine, and push-dose epinephrine. Patients placed on anesthetics for seizure control should be placed on continuous EEG monitoring to titrate the dose of the anesthetic infusions to ensure seizure activity is controlled.

Ketamine infusions have gained popularity over the past decade as a treatment of refractory SE. Ketamine's mechanism of action is significantly different from other antiseizure medications; ketamine is a noncompetitive anti-NMDA antagonist, whereas most other antiseizure medications work on potentiating the effect of GABA. This particular feature makes it an attractive option when pharmacoresistance has started to occur. In addition to targeting a different receptor (and therefore increasing the chances of success), ketamine does not carry the same risk of hypotension or respiratory depression observed with other anesthetics. More recently, ketamine has been proposed as a second-line rescue agent in the out-of-hospital setting, given the challenges associated with administering traditional second-line medications in that setting. It is administered as a bolus or short infusion for over 2 to 3 min, often without securing an airway.[52] In one study, patients who continued to seize despite an adequate dose of midazolam were given 100 mg Ketamine as IV, IM, IO, or IN. Motor seizures were terminated in 98.2% without recurrence in the prehospital or hospital, or any clinically significant complications attributable to ketamine.[53]

A summary of the stepwise approach to SE is summarized in **Fig. 1**. A list of the medication with dosages is in **Table 1**.

Supportive Care

In addition to resuscitation, addressing the underlying cause, and seizure-targeted therapy, patients presenting with SE will require supportive care to further decrease their mortality and morbidity. Providing a safe environment is essential; ensuring the bedrails are in use, in addition to commercial bed rail protectors/pads to help prevent falls and iatrogenic injuries. Bite blocks are not recommended. Patients with SE may develop significant rhabdomyolysis, kidney injury, and lactic acidosis. All these complications may be managed with balanced IV crystalloids to ensure adequate urinary output. Hyperthermia may occur as well and can be managed with external cooling.

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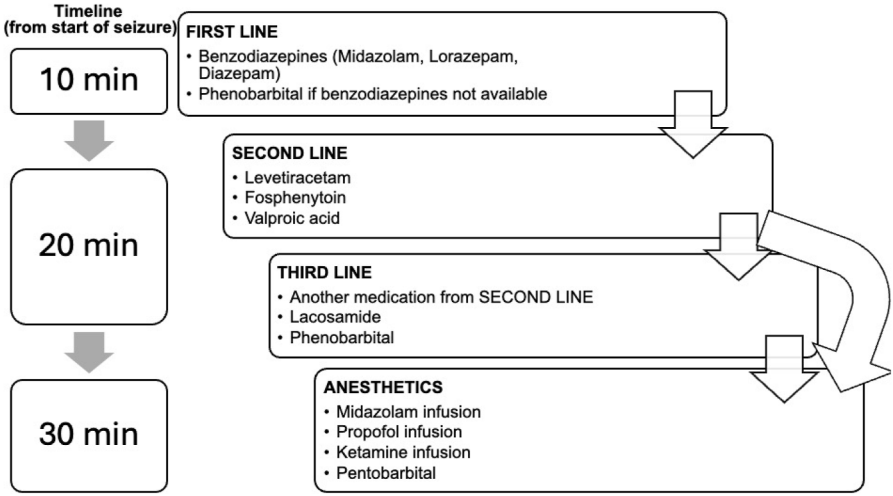

Fig. 1. Stepwise approach to SE.

NONCONVULSIVE STATUS EPILEPTICUS

As described earlier, SE presentation can range from prominent motor symptoms to completely nonconvulsive. NCSE can vary in its presentation, ranging from more subtle symptoms such as confusion and agitation to comatose status. It may also be referred to as subclinical or electrographic-only seizures.[8] NCSE can also initially present as convulsive SE, with 33% to 46% of patients initially in convulsive SE. Of patients with NCSE, 20% to 31% progress to refractory SE, and 11% to 17% progress to super refractory SE.[54,55] NCSE is the most common semiology of SE among patients in the Intensive Care Unit (ICU) and represents a common finding among ICU patients who are comatose or with altered mental status.[56–59] Notably, NCSE was detected in 5% of patients presenting to the ED with altered mental status.[60] Other signs and symptoms that should raise suspicion for NCSE include abnormal eye movements, automatisms or rhythmic activity, staring spells, speech disturbances, or a prolonged postictal period.[61,62] NCSE is diagnosed by EEG.[63]

The variable presentations of NCSE make early specialist consultation with a neurologist and utilization of continuous EEG crucial in its diagnosis and management. All intubated patients initially presenting with SE should be placed on continuous EEG monitoring to assess for NCSE, especially if they were started on anesthetic infusions. Additionally, all patients who are not displaying clear signs of improvement in their alertness within 10 minutes or with impaired consciousness for more than 30 minutes after cessation of seizure activity should be started on continuous EEG.[63] In settings where continuous EEG is not available, intermittent routine EEGs may be used, noting that at least 10% of NCSE may be missed using this method.[64] Limited montage EEGs, which use fewer electrodes compared to standard full montage EEGs, have been found to have comparable sensitivity and specificity for detecting seizure activity with certain configurations of electrodes, representing a viable alternative to full montage EEGs when the latter are unavailable.[65]

DISPOSITION

Patients in SE requiring a second-line agent will likely require admission to the hospital for further management of SE and treatment of the underlying cause, if present. Patients

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who require IV anesthesia and intubation will require admission to the ICU, ideally one with neurocritical care intensivists and continuous EEG monitoring capability.

SPECIAL POPULATIONS

Immunocompromised Patients

Patients with Human immunodeficiency virus/acquired immunodeficiency syndrome (HIV/AIDS) can have numerous etiologies of SE not frequently seen in other populations, including mass lesions, CNS infection from organisms such as *Cryptococcus neoformans*, neurosyphilis, or herpes zoster virus, or toxoplasmosis. These patients require extensive workups for the etiologies of their seizures including maintaining a low threshold for brain imaging and cerebrospinal fluid studies. Phenytoin, carbamazepine, and valproic acid are relatively contraindicated due to their strong induction of the cytochrome P450 system which may interact with HIV/AIDS patients' antiretroviral therapies. Nonenzyme-inducing antiseizure medications such as levetiracetam may be preferred in these patients.[66]

The extensive workup is also necessary for other immunocompromised patients, such as those on chemotherapy or immunosuppressants.

Pregnant and Postpartum Patients

SE can occur in pregnant patients due to a preexisting or new nonpregnancy-related seizure disorder or secondary to a condition precipitated by the pregnancy (such as cerebral venous sinus thrombosis, posterior reversible encephalopathy syndrome, reversible cerebral vasoconstriction syndrome, or eclampsia), and it requires consideration of the wellbeing of both the mother and the fetus.

Status epilepticus due to nonpregnancy-related seizure disorder

Metabolic changes associated with pregnancy can alter the pharmacokinetics of antiseizure medications leading to seizures despite medication adherence.[67] Initial treatment of SE in pregnancy is the same as in nonpregnant individuals. Valproate should be avoided in pregnant patients in SE within the first trimester who are seizing out of concern for teratogenicity, as well as phenobarbital and phenytoin. Levetiracetam appears to have the lowest risk of congenital malformations.[68]

Eclampsia

Eclampsia is defined a convulsive seizure activity unrelated to other medical conditions in pregnant patients usually beyond 20 gestational weeks (and up to 6 weeks postpartum) and in the setting of hypertensive blood pressure readings or evidence of end-organ dysfunction. Up to one-third of patients with eclampsia will have seizures as their first presentation, with no preceding elevated blood pressure, proteinuria, or signs of end-organ damage.[69] Magnesium sulfate infusion, given at 4 to 6 g over 15 min followed by 2 to 3 g/h infusion, is superior to diazepam or phenytoin in controlling seizures due to eclampsia.[70] Magnesium was associated with a decrease in maternal death and seizure recurrence when compared to diazepam.[71] In addition to magnesium sulfate infusion and emergent obstetrics consultation, the patient in SE due to presumed eclampsia should be treated like other patients with SE, with a focus on resuscitation, airway management, and exclusion of other causes. Elevated blood pressure should be treated with IV hydralazine or labetalol. Delivery is the treatment of choice for antepartum patients irrelevant of the gestational age.[69] Patients receiving magnesium should be monitored for signs of magnesium toxicity including hypothermia, hypotension, flaccid paralysis, respiratory depression, and decreased urinary output. Magnesium is contraindicated

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in patients with myasthenia gravis, hypocalcemia, renal failure, cardiac ischemia, heart block, or myocarditis.[69]

Older adults

Among individuals over the age of 60, the incidence of SE is 2 to 5 times greater than that of young adults, and increases with the increase in age.[72] The most common cause of SE in individuals over the age of 60 is a stroke, representing 52.3% of cases.[73] Other important causes in this population include traumatic brain injury, withdrawal from antiseizure medications, CNS infection, or metabolic derangements. SE carries a higher mortality in geriatric patients, with a mortality of 38% in patients 60 to 79 years of age and up to 50% in individuals 80 years and older. This high mortality may be due to changes associated with the aging brain as well as the high prevalence of comorbidities. While the general management of SE remains essentially unchanged compared to younger population,[47] clinicians should thoroughly assess geriatric patients in SE for stroke and other secondary etiologies more commonly found in this patient population.[73]

SUMMARY

SE and its associated morbidity and mortality are potentially preventable with early identification and treatment. Prioritizing resuscitation (especially airway management), rapid administration of appropriately-dosed BZD, adequate utilization of weight-based dosing of second-line antiseizure medications, and escalation to anesthetics when needed form the basis of evidence-based treatment of SE. Creating local protocols facilitate care and improve outcomes.

CLINICS CARE POINTS

- Recognition of status epilepticus is crucial and should be followed immediately by appropriately dosed benzodiazepines
- All patients with seizure activity should be assessed for hypoglycemia, and all female patients of child-bearing potential should have a pregnancy test obtained
- Clinicians should have a high index of suspicion for nonconvulsive status epilepticus. All intubated patients should be started on continuous electroencephalography

DISCLOSURES

The authors have nothing to disclose.

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